The Truth About Cholesterol and Other Misunderstood Nutrients

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50% of all heart attack patients have normal cholesterol levels


The Truth About Cholesterol

• Cholesterol does not cause cardiovascular disease (CVD). High cholesterol is only a risk factor in some people (males age 30-60) of developing heart disease. High cholesterol does not seem to be a risk factor for CVD in women or people over 70.
• Blaming cholesterol for CVD is like blaming firemen for the fire!
The Truth About Cholesterol

- A low fat, low-cholesterol diet will NOT reduce cholesterol levels
- The best way to lower cholesterol is by lowering insulin levels
- At the turn of the last century (1900’s) heart disease was rare. At this time people are eating LOTS of saturated fat...but very little sugar

Metabolic Syndrome

- Obesity (abdominal fat)
- Hypertension
- Glucose intolerance
- High triglycerides
- Low HDL-cholesterol

Insulin resistance is the common denominator to all these conditions
Insulin Resistance

- Insulin acts as the key that unlocks the cells of the body allowing glucose into the cell
- When insulin levels are high too often many cells stop responding, except abdominal fat cells
- Insulin resistance is a plague of the 21st century that underlies obesity, diabetes, high cholesterol and metabolic syndrome (syndrome X)

Cholesterol Review: Lipoproteins

- Lipoproteins (e.g. HDL, LDL) are carriers for cholesterol
- HDL picks up cholesterol from tissues and carries it to the liver for recycling and elimination
- LDL carries cholesterol to the rest of the body for delivery to the tissues
- When LDL becomes oxidized, it can deposit cholesterol in the arteries

Cholesterol Review

- For people over 30 high total cholesterol is defined as over 5.2 mm/L (200 mg/dL for Americans)
- Triglycerides (TG) are another type of fat that are tested at the same time as cholesterol. TG make the blood more likely to clot and can lower the levels of HDL. They are a better indicator of CVD risk than cholesterol.
Better Tests for CVD Risk

- C-reactive protein
- Homocysteine
- Insulin resistance (waistline measurement)
- Lipoprotein(a) (marker for oxidized LDL)
- Triglycerides
- *Coronary Artery Calcium Score

Coronary Artery Calcification (CAC)

- A high CAC score on electron beam tomography has been found to be a better predictor of mortality than age
- Sudden death from heart attack is much more highly correlated with calcification of the aorta than cholesterol

Coronary Artery Calcification (CAC)

- http://www.heartscore.net
- Buffalo MRI
- Need referral from physician, can print out Rx form online
The Importance of the LDL Receptor to Heart Disease

- Evidence from genetic mutations strongly suggests that poor functioning of the LDL receptor is one of the most important causes of heart disease.
- Familial hypercholesterolemia results from a defective LDL receptor: cells cannot take up LDL from the blood. The LDL accumulates and over time it oxidizes.

The Truth About Cholesterol

- We need cholesterol to
  - Make vitamin D
  - Make hormones
  - Make bile
  - Protect us from infection
  - Maintain memory and mental function
  - And much more...

Check out www.cholesterol-and-health.com

Cholesterol and the Brain

- The brain is over 20% cholesterol
- 25% of the body’s cholesterol is in the brain
- Cholesterol is required to form new synapses that are essential for learning and memory
Goals

- Lower insulin resistance and inflammation
- Raise HDL
- Protect from LDL oxidation by ↑ antioxidants and ↓ PUFA intake
- Increase LDL receptor function
- Decrease coronary artery calcification

Insulin Resistance and Viscous Polysaccharides

Niacin

• As effective as statin meds, but unlike statins, niacin actually helps to increase HDL
• Begin with 500 mg with evening meal, increase to 1500 mg over 4 week period. If after a month on this dose there isn’t significant change, then can increase to 3000 mg.
• Can use inositol hexaniacinate to avoid flush but not niacinimide.

Lecithin

• Lecithin in a complex of phospholipids, including choline
• Lecithin help cholesterol connect to lipoproteins (by a magnesium dependant enzyme LCAT), which helps expel cholesterol from the body’s tissues
• Since niacin treatment can deplete choline it is helpful to take lecithin with niacin

Coenzyme Q10

Co Q10 is crucial for electron transport in the mitochondria
Heart cells have a very need for co Q10
 Statins block the production of coQ10, increasing risk of congestive heart failure
Co Q10 and vitamin E are the principal antioxidants in cell membranes and lipoproteins
Curcumin

- A component of turmeric, recent studies show that curcumin increases the expression of LDL receptors

Wheat Free for Heart Health?

- American cardiologist William Davis recommends all patients go wheat free for heart health
- http://www.heartsanblog.org/2008/05/wheat-free-is-not-gluten-free.html

Vitamin K₁

- Aka phylloquinone (synthetic = phytonadione)
- Found in chloroplast membrane
- Vitamin K₁ forms a bridge between chlorophyll and several iron-sulphur centres, across which electrons travel
- Discovered by German scientists as essential for “koagulation”, it is as a cofactor in the formation of coagulation factors II (prothrombin), VII, IX and X by the liver.
Vitamin K1: Phylloquinone

* = vitamin K₁, dependent protein

Coagulation Cascade

- Unlike other fat-soluble vitamins, the body does not store vitamin K
- However, the vitamin K cycle allows a small amount of vitamin K₁ to be recycled many times, minimizing the dietary requirement
- Best dietary source of vitamin K₁ is green leafy veggies
Vitamin K Cycle

Non-Bleeding Side Effects of Long-Term Warfarin Therapy

- "The Calcium Paradox"
- Osteoporosis
- "Significant increases in tissue calcification in [...]heart valves, aorta, coronary and carotid arteries and other areas in body"
  - Donovan JL et al., "Long-Term Warfarin Therapy is Associated with Tissue Calcification: Influence of Treatment Duration, Age, and Gender," Circulation, 2006; 114: II-30

The K Vitamins: Major Misconceptions

- Vitamin K exists primarily in two forms K$_1$ and K$_2$
- Both K vitamins were discovered in the 1930’s
- However, three major misconceptions persisted for 60 years
  - K$_1$ and K$_2$ are merely different forms of the same vitamin (wrong)
  - Blood clotting is their only role (really wrong)
  - Deficiency is rare and obvious
Vitamin K: Beyond Coagulation

- The first K-dependent protein of skeletal metabolism wasn’t discovered until 1978
- Only in 1997 was it recognized that vitamin K plays a major role in health other than blood clotting
- Even more recent studies (2007) show that
  - It is possible to have a clinically-significant vitamin K deficiency and without a bleeding disorder
  - Deficiency is very widespread

Vitamin K₂: the menaquinones

- When animals consume K₁, some of it is converted in tissues to K₂
- The ability to make this conversion varies widely between species
- The ability to convert K₁ to K₂ in humans has not been determined, but seems to be very weak: humans are meant to get most of their K₂ through diet
- Vitamin K₂ can also be made by lactic bacteria
Vitamin K₂

- Unlike the other fat-soluble nutrients (A, D, E) vitamin K₂ is not stored in the body
- There is no recycling of K₂, so it must be provided daily. Despite vitamin K₂’s production by healthy intestinal bacteria, humans can develop a deficiency of the vitamin in as few as 7 days on a vitamin K₂-deficient diet.


The Work of Weston A. Price, D.D.S.

- In the 1930s, Dr. Price travelled the globe to study the health of populations untouched by western civilization
- His studies revealed that dental caries and deformed dental arches resulting in crowded, crooked teeth are the result of nutritional deficiencies, not inherited genetic defects
- His work revealed a strong connection between other health problems – even social and learning problems – and diet

- Price discovered an additional fat-soluble nutrient (“Activator X”) that is present in fish livers, shellfish and dairy fat from cows
- All primitive groups studied had a source of Activator X in their diets
The Factor X Mystery Solved

- In the 1940’s Dr. Royal Lee suggested that Activator X was essential fatty acids
  - Lee, R., “Butter, Vitamin E and the “X” Factor of Dr. Price,” In Dr. Royal Lee Historical Archive Collection from Selene River Press, Not Date.
  - http://www.seleneriverpress.com
- In 1980 Dr. Jeffrey Bland suggested that it was EPA
- Only in 2007 was Activator X definitively shown to be vitamin K₂

Gamma-carboxylation

- Vitamin K is the cofactor for the enzyme, γ-glutamyl carboxylase
- This converts glutamic acid residues in a number of substrate proteins to γ-carboxy- glutamic acid (Gla) residues, which then serve to form calcium-binding groups in these proteins and are essential for their biologic activity

Vitamin K-Dependent Carboxylation

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Ontario Association of Naturopathic Doctors Continuing Education
The Truth About Cholesterol and Other Misunderstand Nutrients
with Kate Rhéaume-Bleue, BSc, ND
Tuesday, March 8, 2011
Gamma-carboxylation

- Vitamin K-induced carboxylation thus activates this family of Gla-proteins, which are involved in numerous essential activities throughout the body, including blood coagulation, bone metabolism, vascular repair, prevention of vascular calcification, regulation of cell proliferation (i.e. cancer prevention), and signal transduction.

- $\text{K}_1$ is preferentially utilized in the carboxylation of clotting factors in the liver.
- $\text{K}_2$ is preferentially used in the rest of the body to carboxylate the other vitamin K-dependent Gla-proteins, including osteocalcin, and matrix-Gla protein (MGP) which prevents calcification of soft tissue and is primarily synthesized in cartilage and vessel wall.
- Undercarboxylated Gla proteins will produce the Calcium Paradox.

Clinical effects of $\text{K}_2$ deficiency/undercarboxylation of Gla proteins

- Osteoporosis
- Atherosclerosis: calcification of aorta, coronary arteries, etc.
- Dental caries, crowded teeth in offspring
- Varicosities
- Insulin resistance
- Cancer (prostate and liver)
Vitamin K<sub>2</sub> and Bone Health

- There are at least three Gla proteins associated with bone tissue, of which osteocalcin is the most abundant and best known.
- Only after its carboxylation by K<sub>2</sub> is osteocalcin able to attract calcium ions and incorporate them into hydroxyapatite crystals forming the bone matrix.
- When vitamin K<sub>2</sub> levels are insufficient, osteocalcin remains uncarboxylated and bone mineralization is impaired.

Vitamin K<sub>2</sub> and Bone Health

- K<sub>2</sub> also inhibits osteoclast differentiation.
- This is necessary to benefit from bone-building effects of vitamin D<sub>3</sub>'s upregulation of osteocalcin.

Vitamin K<sub>2</sub> and Bone Health

- Supplementing with K<sub>2</sub> increases bone density and reduces fracture risk.
- Clinical trials have shown that the combination of K<sub>2</sub> and vitamin D<sub>3</sub> is more effective in preventing bone loss than either nutrient alone.
Calcium and Heart Health

• “Healthy older women randomised to calcium supplementation showed increased rates of [atherosclerosis and] myocardial infarction. This effect could outweigh any benefits on bone from calcium supplements”

Coronary Artery Calcification (CAC)

• A high CAC score on electron beam tomography has been found to be a better predictor of mortality than age

• Sudden death from heart attack is much more highly correlated with calcification of the aorta than cholesterol

OACs and CAC

• "Matrix-carboxyglutamic acid (Gla) protein (MGP) is a potent inhibitor of vascular calcification, the activity of which is regulated by vitamin K. In animal models, vitamin K antagonists (oral anticoagulants [OACs]) were shown to induce arterial calcification... Calcifications in patients receiving preoperative OAC treatment were significantly (2-fold) larger than in nontreated patients. These observations suggest that OACs, which are widely used for antithrombotic therapy, may induce cardiovascular calcifications as an adverse side effect.”
ucMGP and CAC

- Serum uncarboxylated MGP levels are inversely correlated with the severity of CAC

MGP and CAC

- Matrix Gla-protein (MGP) is the strongest inhibitor of tissue calcification presently known
- MGP is produced by small muscle cells in the vasculature where – once carboxylated by K₂ – it protects against calcification through several mechanisms
  - Pizzorno L., "Vitamin D and Vitamin K Team Up to Lower CVD Risk," Longevity Medicine Review online ref

Vitamin K₂ and Heart Disease

- "The risk of incident CHD, all-cause mortality, and aortic atherosclerosis was studied in tertiles of energy-adjusted vitamin K intake after adjustment for age, gender, BMI, smoking, diabetes, education, and dietary factors. Phylloquinone intake was not related to any of the outcomes. These findings suggest that an adequate intake of menaquinone could be important for CHD prevention."
Vitamin K₂ and Varicosities

- “Undercarboxylated MGP is prevalent in varicose veins compared to healthy veins. Smooth muscle cells from varicose veins show enhanced matrix mineralization. MGP in varicose veins may contribute to venous wall remodeling by affecting proliferation and mineralization processes probably through impaired carboxylation of MGP.”

Vitamin K₂ and Metabolic Syndrome

- “Mice lacking the osteoblast-secreted molecule osteocalcin display decreased cell proliferation, glucose intolerance, and insulin resistance. Ex vivo, osteocalcin can stimulate insulin expression in cells and Adiponectin, an insulin-sensitizing adipokine, in adipocytes; in vivo osteocalcin can improve glucose tolerance... The skeleton thus exerts an endocrine regulation of sugar homeostasis...”

Vitamin D and Metabolic Syndrome

- Evidence shows a strong relationship between serum concentrations of 25(OH)D and HDL-C concentrations: each 10 ng/mL increment in 25(OH)D was associated with an increase of 3.8 to 4.2 mg/dL in HDL-C
- Also inverse associations between vitamin D levels and triglyceride levels, body mass index, and waist circumference
K₂ and Menopause

- Estrogen is involved in the conversion of vitamin D to its active bone-building form (1,25-dihydroxycholecalciferol [1,25(OH)₂D] or calcitriol)
- When estrogen levels drop, osteoclasts become more sensitive to parathyroid hormone, increasing their activity
- The decline in estrogen also increase production of IL-6 which stimulates osteoclast production

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K₂ and Menopause

- Supplementing with K2 (MK-7) has been shown to compensate for the changes in bone density due to menopause

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K₂ and Menopause

- "Vitamin K₂ binds 17β-hydroxysteroid dehydrogenase 4 and decreases the intracellular estradiol:estrone ratio, which resulted in the inhibition of the amount of estrogen receptor α-binding to its target DNA. These results suggest a possible novel role for vitamin K in modulating estrogen function"
Interactions Between Vitamins A, D and K$_2$

- Osteocalcin is responsible for organizing calcium deposition in bones and teeth
- Cells only produce this protein in the presence of both vitamins A and D
- However, osteocalcin will only facilitate the deposition of calcium salts once it has been activated by vitamin K$_2$

Interactions Between Vitamins A, D and K$_2$

- Vitamins A and D regulate the expression of matrix GlA (MGP) which is also responsible for mineralizing bone and protecting arteries from calcification
- Like osteocalcin, MGP can only fulfill its function once activated by vitamin K$_2$
Interactions Between Vitamins A, D and K<sub>2</sub>

- Vitamin D upregulates the expression of Gla-proteins, whose activation depends on vitamin K-mediated carboxylation.
- Vitamin D thus increases both the demand for vitamin K and the potential for benefit from K-dependent proteins, including osteocalcin in bone and MGP in blood vessels.


Interactions Between Vitamins A, D and K<sub>2</sub>

- If the production of vitamin K-dependent proteins exceeds the capacity of the vitamin K pool or the enzyme that uses vitamin K, the vitamin K carboxylase, to activate them, these proteins will be undercarboxylated (defective).

Interactions Between Vitamins A, D and K<sub>2</sub>

- Increased levels of vitamin D may actually induce a functional vitamin K<sub>2</sub> deficiency, with the result that levels of uncarboxylated osteocalcin and matrix-Gla protein rise in the circulation and vasculature. In this case, not only is calcium not delivered to the bones, but it is deposited in the arteries and soft tissues, which become calcified.

Interactions Between Vitamins A, D and K₂

- Vitamin D causes a large increase in the production of MGP
- When vitamin A is added in combination with vitamin D the former downregulates MGP production to normal levels
- Vitamin A thus protects against possible vitamin D toxicity by exerting a vitamin K-sparing effect


Interactions Between Vitamins A, D and K₂

- Warfarin, (induces a functional vitamin K deficiency) has definitively been shown to produce extensive hypervitaminosis D-like calcification of the soft tissues
- This toxicity increases synergistically with vitamin D when the two are combined
- Will retinol help?

Assessing Vitamin D Status

- The body’s ability to utilize cholecalciferol is not optimized until blood levels of 25(OH)D are ≥40 ng/ml (98 nmol/L)

Assessing Vitamin K Status

- A ucOC (uncarboxylated osteocalcin) test is the best measure of vitamin K status.
- High levels of uncarboxylated osteocalcin indicate insufficient vitamin K for bone health and indirectly indicate that MGP is insufficiently carboxylated.
- http://www.metametrix.com/

Toxicity of Liposoluble Vitamins

- Adequate Vitamin D protects against vitamin A toxicity.
- Adequate vitamin A protects against D toxicity.
- “An extreme imbalance between vitamins A and D leads to the synthesis of abnormally high amounts of MGP. If there is enough vitamin K to activate all of the MGP, it will help protect the soft tissues from calcification. If not, soft tissue calcification ensues.”

Dietary Vitamin A

- Vegetable foods do not contain retinol.
- The conversion of β-carotene to retinol varies widely between individuals, ranging from 0 to 50% conversion.
- Diabetes, hypothyroid, excessive consumption of polyunsaturated fatty acids, zinc deficiency and even cold weather can hinder the conversion of carotenes to vitamin A.
### Dietary Vitamin A

<table>
<thead>
<tr>
<th>Food Source of Retinol</th>
<th>Vitamin A (IU)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver, beef, cooked 3 oz.</td>
<td>30,325</td>
</tr>
<tr>
<td>Liver, chicken, cooked, 3 oz.</td>
<td>13,920</td>
</tr>
<tr>
<td>Egg substitute, fortified, ¼ cup</td>
<td>1,355</td>
</tr>
<tr>
<td>Fat-free milk, fortified with vitamin A, ½ cup</td>
<td>500</td>
</tr>
<tr>
<td>Milk, whole, 3.25% fat, 2 cup</td>
<td>305</td>
</tr>
<tr>
<td>Cheddar cheese, 1 oz</td>
<td>300</td>
</tr>
<tr>
<td>Whole egg, 1 medium</td>
<td>280</td>
</tr>
</tbody>
</table>

Source: NIH Office of Dietary Supplements

### Vitamin K₂ in the Diet: What went wrong?

- Vitamin K₂ has become almost impossible to get in the modern diet due to the advent of industrial farming techniques and confined animal feeding operations
- K₂ is found in dairy products, egg yolks and organ meats, etc. of animals that feed on GRASS – not grain
- The way we produce food has drastically decreased vitamin K₂ in our diet

### K₂ in Clinical Practice

- Diet: choosing traditionally-raised, grass-fed animal products (not just organic)
- Diet: including more fermented foods, natto in particular
- Supplementation
Increasing K₂ in the Diet

- Vitamin K₂ is directly associated with both chlorophyll and beta-carotene within a single protein complex and plays a direct role in photosynthesis.
- The colour of grass and its rate of growth directly indicate the vitamin K₁ content of their diet.
- Animals grazing on grass will accumulate vitamin K₂ in their tissue in direct proportion to vitamin K₁ content of their diet.

Increasing K₂ in the Diet

- Choosing grass-fed meat, eggs and dairy products will increase dietary K₂ intake.
- A supplement will still be necessary in the winter months, if not year round.

Vitamin K₂ in Foods (MCG/100g)

<table>
<thead>
<tr>
<th>Food</th>
<th>Vitamin K₂ (MCG/100g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Natto</td>
<td>1103.4 (100% MK-7)</td>
</tr>
<tr>
<td>Goose Liver</td>
<td>369.0 (100% MK-4)</td>
</tr>
<tr>
<td>Hard Cheeses</td>
<td>76.3 (0% MK-4)</td>
</tr>
<tr>
<td>Soft Cheeses</td>
<td>35.5 (0.5% MK-4)</td>
</tr>
<tr>
<td>Egg Yolk (Netherlands)</td>
<td>32.1 (0% MK-4)</td>
</tr>
<tr>
<td>Goose Leg</td>
<td>31.0 (100% MK-4)</td>
</tr>
<tr>
<td>Egg Yolk (US)</td>
<td>15.5 (100% MK-4)</td>
</tr>
<tr>
<td>Butter</td>
<td>15.0 (100% MK-4)</td>
</tr>
<tr>
<td>Chicken Liver</td>
<td>14.1 (100% MK-4)</td>
</tr>
<tr>
<td>Chicken Breast</td>
<td>8.9 (100% MK-4)</td>
</tr>
<tr>
<td>Ground Beef</td>
<td>8.1 (100% MK-4)</td>
</tr>
<tr>
<td>Bacon</td>
<td>5.6 (100% MK-4)</td>
</tr>
<tr>
<td>Calf Liver</td>
<td>5.0 (100% MK-4)</td>
</tr>
<tr>
<td>Sauerkraut</td>
<td>4.8 (0% MK-4)</td>
</tr>
<tr>
<td>Whole Milk</td>
<td>1.0 (100% MK-4)</td>
</tr>
<tr>
<td>Salmon</td>
<td>0.3 (100% MK-4)</td>
</tr>
<tr>
<td>Egg White</td>
<td>0.4 (100% MK-4)</td>
</tr>
<tr>
<td>Skim Milk</td>
<td>0.0</td>
</tr>
</tbody>
</table>
RDI and ODI

- Current RDI are inadequate and do not distinguish between \( K_1 \) and \( K_2 \)
- Optimal daily intake is being established
- Vitamin \( K_2 \) requirements increase with age

Natto

- A traditional Japanese food made from soybeans fermented by *Bacillus subtilis*
- A breakfast food eaten with rice in some (but not all) areas of Japan
- Excellent source of \( K_2 \), in the form of MK-7

- Natto contains several other compounds that are health promoting
- Pyrazine is a heterocyclic aromatic ring that gives natto its distinct smell and reduces the likelihood of blood clotting ref
Natto

- Nattokinase is a serine protease type enzyme that may also reduce blood clotting both by direct fibrinolysis of clots, and inhibition of the plasma protein plasminogen activator inhibitor-1. This may help to avoid thrombosis.
- Studies have shown that oral administration of nattokinase in enteric capsules leads to a mild enhancement of fibrinolytic activity in rats and dogs, but there have been no human clinical trials published as of 08/2009.

Natto

- “Serum MK-7 and γ-carboxylated osteocalcin concentrations in men with the occasional or frequent dietary intake of natto were significantly higher than those without any intake. The present study suggests that intake of fermented soybean (natto) increases serum levels of MK-7 and γ-carboxylated osteocalcin.”

Natto

- “Natto is eaten frequently in eastern (Tokyo) but seldom in western (Hiroshima) Japan. Serum concentrations of MK-7 is significantly higher in frequent natto eaters. A statistically significant inverse correlation was found between incidence of hip fractures in women and natto consumption in each prefecture throughout Japan.”
**K₂ in Clinical Practice**

- Health Canada has only made vitamin K products available without a prescription since October 2005.
- Vitamin K was previously listed in Part II of Schedule F of the *Food and Drug Regulations*, it is now on Schedule 1 of the *Natural Health Products Regulations*.


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**K₂ in Clinical Practice**

- Health Canada has limited the total amount of vitamin K (1 or 2) to 120 mcg per daily dose.

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**K₂ in Clinical Practice**

- There are two commercially-available forms of K₂ that they differ in clinically-significant ways.
- MK-4 is a short-chain menaquinone available as a synthetic compound (menatetrenone) from tobacco extract.
- MK-7, a long chain menaquinone, is naturally derived from natto fermentation.
K₂ in Clinical Practice

- The majority of the research been done using synthetic MK-4 at doses of 45 mg per day (typically 15 mg tid due to short half-life)
- This is way beyond the daily dosage for supplements available in Canada

MK-7 in Clinical Practice

- MK-7 is highly bioavailable and bioactive: only 45 mcg/day is sufficient to activate osteocalcin
- MK-7 has serum half-life of 3 days – enables the body to build up a reserve that can continuously supply vitamin K₂ to all tissues


K₂ in Clinical Practice

- In patients on OAC therapy, providing up to 50 mcg of MK-7 induced more complete carboxylation of osteocalcin without interfering with OAC therapy

K₂ in Clinical Practice

- Vitamin K₂ supplementation protects against coumarin-induced side effects and reduces diet-induced fluctuations in their INR values

Other K₂ Supplements

- “X Factor butter oil” (various brands)
- Should contain high levels of natural MK-4
- Not standardized for K₂ content
Indications for K\textsubscript{2} supplementation

- Osteoporosis
- Atherosclerosis, history of CVD
- Obesity, diabetes, metabolic syndrome
- Varicose veins
- Dental caries
- Prostate cancer
- Viral cirrhosis
- Renal disease
- Oral anti-coagulant therapy
- In infants, children, pregnant, lactating, and menopausal women

Conclusions

- Vitamin K deficiency may be the most important nutritional factor contributing to the major diseases of our day
- This has happened due to industrial farming practice and consumption of processed foods
- A mass movement towards grass-fed animal products and traditional foods is required
- In the meantime, supplementation with all the fat-soluble vitamins is indicated for every patient

Recommended Reading

- Masterjohn Chris, (Everything including Daily Lipid blog)
- Nourishing Traditions Cookbook
Further reading

• The Cholesterol Myths: Exposing the Fallacy That Saturated Fat and Cholesterol Cause Heart Disease by Uffe Ravnskov, MD, PhD (2000).
• The Great Cholesterol Con: Why everything you’ve been told about cholesterol, diet, and heart disease is wrong! by Anthony Colpo (2006).

Further reading

• The Great Cholesterol Con: The Truth About What Really Causes Heart Disease and How to Avoid It by Malcolm Kendrick, MD (2007).
• Lipitor Thief of Memory by Duane Graveline, M.D. (2006)